	FILE 'HCAPLUS' ENTERED AT 15:42:55 ON 14 MAY 2009
L1	90980 S GLYCOSAMINOGLYCAN OR HEPARIN OR HYALURON? OR DERMATAN
L2	819295 S (AMINO ACID) OR LEUCINE OR LYSINE OR CYSTEINE
L3	67430 S ASTHMA OR BRONCHITIS OR (CYSTIC FIBROSIS) OR COPD OR (CHRONIC
L4	126 S L1 AND L2 AND L3
L5	54 S L4 AND (PY<2004 OR AY<2004 OR PRY<2004)
	FILE 'STNGUIDE' ENTERED AT 15:43:30 ON 14 MAY 2009
	FILE 'HCAPLUS' ENTERED AT 15:44:05 ON 14 MAY 2009
L6	25742 S INHALER OR INHALENT OR (DRY POWDER) OR INHALED OR INHALABLE
L7	1 S L5 AND L6
	FILE 'HCAPLUS' ENTERED AT 15:44:57 ON 14 MAY 2009
L8	12603 S MUCUS OR MUCOACTIVE
L9	3 S L5 AND L8
	FILE 'HCAPLUS' ENTERED AT 15:48:17 ON 14 MAY 2009
L10	299147 S LEUCINE OR LYSINE OR CYSTEINE
L11	27 S L5 AND L10

=> file hcaplus
COST IN U.S. DOLLARS

FULL ESTIMATED COST

SINCE FILE TOTAL ENTRY SESSION 0.88 0.88

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FILE COVERS 1907 - 14 May 2009 VOL 150 ISS 20 FILE LAST UPDATED: 13 May 2009 (20090513/ED) REVISED CLASS FIELDS (/NCL) LAST RELOADED: Feb 2009 USPTO MANUAL OF CLASSIFICATIONS THESAURUS ISSUE DATE: Feb 2009

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This file contains CAS Registry Numbers for easy and accurate substance identification.

=> s glycosaminoglycan or heparin or hyaluron? or dermatan

12054 GLYCOSAMINOGLYCAN

53788 HEPARIN

32297 HYALURON?

5056 DERMATAN

L1 90980 GLYCOSAMINOGLYCAN OR HEPARIN OR HYALURON? OR DERMATAN

=> s (amino acid) or leucine or lysine or cysteine

1219862 AMINO

4826619 ACID

L2

616011 AMINO ACID

(AMINO(W)ACID)

101829 LEUCINE

120003 LYSINE

119714 CYSTEINE

819295 (AMINO ACID) OR LEUCINE OR LYSINE OR CYSTEINE

 \Rightarrow s asthma or bronchitis or (cystic fibrosis) or COPD or (chronic obstructive pulmonary disease) or bronchiectasis

44127 ASTHMA

8110 BRONCHITIS

19601 CYSTIC

48313 FIBROSIS

14861 CYSTIC FIBROSIS

(CYSTIC(W)FIBROSIS)

4726 COPD

260972 CHRONIC

17024 OBSTRUCTIVE

109125 PULMONARY

1150280 DISEASE

9461 CHRONIC OBSTRUCTIVE PULMONARY DISEASE

(CHRONIC (W) OBSTRUCTIVE (W) PULMONARY (W) DISEASE)

921 BRONCHIECTASIS

L3 67430 ASTHMA OR BRONCHITIS OR (CYSTIC FIBROSIS) OR COPD OR (CHRONIC OBSTRUCTIVE PULMONARY DISEASE) OR BRONCHIECTASIS

=> s 11 and 12 and 13

L4 126 L1 AND L2 AND L3

=> s 14 and (PY<2004 or AY<2004 or PRY<2004)

24035465 PY<2004 4798798 AY<2004 4271441 PRY<2004

L5 54 L4 AND (PY<2004 OR AY<2004 OR PRY<2004)

=> file stnquide

COST IN U.S. DOLLARS

SINCE FILE TOTAL

ENTRY SESSION

FULL ESTIMATED COST

2.85 3.73

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FILE CONTAINS CURRENT INFORMATION.

LAST RELOADED: May 11, 2009 (20090511/UP).

=> file hcaplus

COST IN U.S. DOLLARS

SINCE FILE TOTAL ENTRY SESSION

FULL ESTIMATED COST

0.07 3.80

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FILE COVERS 1907 - 14 May 2009 VOL 150 ISS 20 FILE LAST UPDATED: 13 May 2009 (20090513/ED)

REVISED CLASS FIELDS (/NCL) LAST RELOADED: Feb 2009 USPTO MANUAL OF CLASSIFICATIONS THESAURUS ISSUE DATE: Feb 2009

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=> s inhaler or inhalent or (dry powder) or inhaled or inhalable

2657 INHALER
2 INHALENT
517983 DRY
631737 POWDER

6208 DRY POWDER

(DRY(W)POWDER)

17477 INHALED

1328 INHALABLE

L6 25742 INHALER OR INHALENT OR (DRY POWDER) OR INHALED OR INHALABLE

=> s 15 and 16

=> file stnquide

COST IN U.S. DOLLARS
SINCE FILE TOTAL
ENTRY SESSION
FULL ESTIMATED COST
2.85
6.65

FILE 'STNGUIDE' ENTERED AT 15:44:07 ON 14 MAY 2009 USE IS SUBJECT TO THE TERMS OF YOUR CUSTOMER AGREEMENT COPYRIGHT (C) 2009 AMERICAN CHEMICAL SOCIETY (ACS)

FILE CONTAINS CURRENT INFORMATION.
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=> file hcaplus
COST IN U.S. DOLLARS

COST IN U.S. DOLLARS SINCE FILE TOTAL ENTRY SESSION FULL ESTIMATED COST 0.07 6.72

FILE 'HCAPLUS' ENTERED AT 15:44:57 ON 14 MAY 2009
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This file contains CAS Registry Numbers for easy and accurate substance identification.

=> s mucus or mucoactive

12561 MUCUS 70 MUCOACTIVE

L8 12603 MUCUS OR MUCOACTIVE

=> s 15 and 18

L9 3 L5 AND L8

=> file stnquide

COST IN U.S. DOLLARS

SINCE FILE TOTAL ENTRY SESSION 2.85 9.57

FULL ESTIMATED COST

FILE 'STNGUIDE' ENTERED AT 15:44:59 ON 14 MAY 2009 USE IS SUBJECT TO THE TERMS OF YOUR CUSTOMER AGREEMENT COPYRIGHT (C) 2009 AMERICAN CHEMICAL SOCIETY (ACS)

FILE CONTAINS CURRENT INFORMATION.
LAST RELOADED: May 11, 2009 (20090511/UP).

=> d 19 1-3 ti abs bib YOU HAVE REQUESTED DATA FROM FILE 'HCAPLUS' - CONTINUE? (Y)/N:y

- L9 ANSWER 1 OF 3 HCAPLUS COPYRIGHT 2009 ACS on STN
- TI Methods and compounds for the treatment of mucus hypersecretion by inhibiting mucus secretion using compounds having targeting and translocating modified light chain of clostridial neurotoxin
- AB A method of treating mucus hypersecretion, the causative factor in chronic obstructive pulmonary disease (COPD), asthma and other clin. conditions involving COPD, comprises administering a compound that inhibits exocytosis in mucus secreting cells or neurons that control or direct mucus secretion. Also described is a compound, for use in the treatment of hypersecretion of mucus, which inhibits mucus secretion by inhibiting mucus secretion by mucus secreting cells, and/or inhibiting neurotransmitter release from neuronal cells controlling or directing mucus secretion. The compound comprises: (a) a light chain (L-chain) or L-chain

fragment of a clostridial neurotoxin, which L-chain or L-chain fragment includes the active proteolytic enzyme domain of the L-chain; (b) a targeting domain that binds to a target cell selected from the group consisting of (i) a mucus secreting cell, and (ii) a neuronal cell controlling or directing mucus secretion; and (c) a translocating domain that translocates the L-chain or L-chain fragment into the target cell. Substance P, as the targeting domain, was conjugated to clostridial neurotoxin fragment LHN/A.

- AN 2008:159937 HCAPLUS <<LOGINID::20090514>>
- DN 148:230138
- TI Methods and compounds for the treatment of mucus hypersecretion by inhibiting mucus secretion using compounds having targeting and translocating modified light chain of clostridial neurotoxin
- IN Quinn, Conrad Padraig; Foster, Keith Alan; Chaddock, John
- PA Syntaxin Ltd., UK
- SO U.S. Pat. Appl. Publ., 80pp., Cont.-in-part of U.S. Ser. No. 518,213. CODEN: USXXCO
- DT Patent
- LA English
- FAN.CNT 4

	PATENT NO.)	DATE		API	PLICAT	ION	DA					
ΡI		20080032928				A1 20080207				2007-			<					
		20000				A2			0302	WO	1999-	GB28	06		19	99908	325	<
	WO	20000				A3		2000	0615									
			AU,	,							_	_						
		RW:	AT,	BE,	CH,	CY,	DE,	, DK,	ES,	FI, F	R, GB,	GR,	ΙE,	IT,	LU,	MC,	NL,	
			PT,	SE														
	US	66324	440			В1		2003	1014	US	2001-	7636	69		20	00105	529	<
	US	20040	0071	736		A1		2004	0415	US	2003-	6336	98		20	00308	805	<
	US	20070	010	447		A1		2007	0111	US	2006-	5182	13		20	0609	911	<
	US	20080	02490	019		A1		2008	1009	US	2008-	1017	49		20	0804	411	<
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	WO	1999-	-GB28	306		W		1999	0825	<								
	US	S 2001-763669 A2			2001	0529	<											
	US	2003-	-6336	698		В1		2003	0805	<								
	US	2006-	-5182	213		A2		2006	0911									
	US	2007-	-806	496		A2		2007	0531									

- L9 ANSWER 2 OF 3 HCAPLUS COPYRIGHT 2009 ACS on STN
- TI Pharmaceutical compositions
- AB The present invention relates to pharmaceutical compns. which are useful in the treatment of diseases where excess mucus is present in the respiratory tract, such as cystic fibrosis and chronic obstructive pulmonary disease
 - In particular, the invention relates to pharmaceutical compns. for administration by pulmonary inhalation. Thus, in a first aspect of the present invention, a composition for assisting mucus clearance is provided, the composition comprising one or more mucoactive agents for reducing crosslinking within the mucus; for diluting the mucus; and/or for digesting naked DNA and cell debris within the mucus. Preferably, the composition according to the invention further has the effect of reducing inflammation. In one embodiment of the present invention, the composition comprises one or more mucoactive agents together with an addnl. active agent such as an anti-inflammatory agent. In a particularly preferred embodiment of the present invention, the mucoactive agent for reducing crosslinking is a glycosaminoglycan such as heparin. A further group of mucoactive agents capable of assisting mucus clearance are amino acids. Acetylcysteine (NAC) and the acetylcysteine salt derivative Nacystelyn (or NAL) are also effective mucoactive agents which

2005:259852 HCAPLUS <<LOGINID::20090514>> ΑN 142:329858 DN Pharmaceutical compositions ΤI Morton, David; Ganderton, David; Staniforth, John; Kamlag, Yorick ΙN PAVectura Limited, UK SO PCT Int. Appl., 60 pp. CODEN: PIXXD2 DT Patent English LA FAN.CNT 1 KIND DATE APPLICATION NO. ----WO 2005025540 A2 20050324 WO 2004-GB3932 20040915 <-- WO 2005025540 A3 20050616 РΤ W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG A1 20050324 AU 2004-271778 A1 20050324 CA 2004-2538399 A2 20060607 EP 2004-768478 AU 2004271778 20040915 <--CA 2538399 A1 20040915 <--EP 1663151 20040915 <--R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, PL, SK, HR BR 2004014425 A 20061114 BR 2004-14425 20040915 <--BR 2004014425 A 20061114 BR 2004-14425 20040915 <-CN 1874757 A 20061206 CN 2004-80032679 20040915 <-JP 2007505830 T 20070315 JP 2006-525902 20040915 <-SG 146649 A1 20081030 SG 2008-6902 20040915 <-KR 2006082865 A 20060719 KR 2006-705166 20060314 <-MX 2006002952 A 20060920 MX 2006-2952 20060315 <-ZA 2006002748 A 20070530 ZA 2006-2748 20060404 <-IN 2006CN01269 A 20070629 IN 2006-CN1269 20060413 <-US 20070065373 A1 20070322 US 2006-571184 20060717 <-PRAI GB 2003-27723 A 2003915 <-WO 2004-GB3932 W 20040915

BE.CNT 7 THERE ARE 7 CITED REFERENCES AVAILABLE FOR THIS RECORD RE.CNT 7 THERE ARE 7 CITED REFERENCES AVAILABLE FOR THIS RECORD ALL CITATIONS AVAILABLE IN THE RE FORMAT ANSWER 3 OF 3 HCAPLUS COPYRIGHT 2009 ACS on STN L9 Bronchial mucus hypersecretion in acute quadriplegia. ΤI Macromolecular yields and glycoconjugate composition In acute quadriplegia the authors have noted that about 1 in 5 patients AB develops unexplained production of excessive and tenacious bronchial mucus. Spontaneous recovery from mucus hypersecretion usually occurs within weeks to months. Mucus samples collected from 12 patients were abnormal. Macromol. contents of single aspirates yielded as much as 500 mg. Anal. ultracentrifuge anal. showed the mucus to contain considerable epithelial glycoprotein (GP) of typical buoyant d.; its amino acid and carbohydrate compns. were characteristic of the GP from hypersecretory bronchial mucus such as in chronic bronchitis and cystic fibrosis. In five patients studied after recovery from hypersecretion, there tended to be relatively less GP. The mucus

are suitable for inclusion in the compns. of the present invention.

samples contained a high-d. glycoconjugate (GC): this had sugars of GP but also reacted pos. with a monoclonal antibody to keratan sulfate. Its amino acid composition was different from that of GP: threonine was lower and glycine was higher than in GP. In mucus from one patient who died, chondroitin sulfate ABC and hyaluronic acid were identified as well. This suggests proteoglycans are involved in the pathophysiol. of mucus hypersecretion. The sudden onset and spontaneous recovery of hypersecretion suggests that it is not due to gland hypertrophy. The authors speculate that in acute quadriplegia it is due to disturbed neuronal control of bronchial mucous gland secretion, perhaps related to initial disappearance and later reappearance of peripheral sympathetic nervous system tone. 1991:245405 HCAPLUS <<LOGINID::20090514>> 114:245405 OREF 114:41397a,41400a Bronchial mucus hypersecretion in acute quadriplegia. Macromolecular yields and glycoconjugate composition Bhaskar, K. Ramakrishnan; Brown, Robert; O'Sullivan, Donna Defeudis; Melia, Stephen; Duggan, Marie; Reid, Lynne Dep. Pathol., Child. Hosp., Boston, MA, USA American Review of Respiratory Disease (1991), 143(3), 640-8 CODEN: ARDSBL; ISSN: 0003-0805 Journal

=> d his

English

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CS

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DT

LA

L5

(FILE 'HOME' ENTERED AT 15:40:41 ON 14 MAY 2009)

FILE 'HCAPLUS' ENTERED AT 15:42:55 ON 14 MAY 2009 90980 S GLYCOSAMINOGLYCAN OR HEPARIN OR HYALURON? OR DERMATAN L1819295 S (AMINO ACID) OR LEUCINE OR LYSINE OR CYSTEINE L2 L3 67430 S ASTHMA OR BRONCHITIS OR (CYSTIC FIBROSIS) OR COPD OR (CHRONIC L4126 S L1 AND L2 AND L3

54 S L4 AND (PY<2004 OR AY<2004 OR PRY<2004)

FILE 'STNGUIDE' ENTERED AT 15:43:30 ON 14 MAY 2009

FILE 'HCAPLUS' ENTERED AT 15:44:05 ON 14 MAY 2009 L6 25742 S INHALER OR INHALENT OR (DRY POWDER) OR INHALED OR INHALABLE L7 1 S L5 AND L6

FILE 'STNGUIDE' ENTERED AT 15:44:07 ON 14 MAY 2009

FILE 'HCAPLUS' ENTERED AT 15:44:57 ON 14 MAY 2009 12603 S MUCUS OR MUCOACTIVE $\Gamma8$

3 S L5 AND L8 L9

FILE 'STNGUIDE' ENTERED AT 15:44:59 ON 14 MAY 2009

FILE 'HCAPLUS' ENTERED AT 15:45:12 ON 14 MAY 2009

FILE 'STNGUIDE' ENTERED AT 15:45:12 ON 14 MAY 2009

=> log hold COST IN U.S. DOLLARS SINCE FILE TOTAL ENTRY SESSION FULL ESTIMATED COST 0.07 21.56 DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS) SINCE FILE TOTAL ENTRY SESSION 0.00 -2.46

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STN INTERNATIONAL SESSION SUSPENDED AT 15:45:17 ON 14 MAY 2009

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FULL ESTIMATED COST	0.07	21.56
DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE ENTRY	TOTAL SESSION
CA SUBSCRIBER PRICE	0.00	-2.46
=> file hcaplus COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	0.07	21.56
DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE ENTRY	TOTAL SESSION
CA SUBSCRIBER PRICE	0.00	-2.46

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=> s leucine or lysine or cysteine

101829 LEUCINE 120003 LYSINE 119714 CYSTEINE

L10 299147 LEUCINE OR LYSINE OR CYSTEINE

=> s 15 and 110

L11 27 L5 AND L10

=> file stnquide

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=> d l11 1-27 ti YOU HAVE REQUESTED DATA FROM FILE 'HCAPLUS' - CONTINUE? (Y)/N:y

- L11 ANSWER 1 OF 27 HCAPLUS COPYRIGHT 2009 ACS on STN
- TI Pharmaceutical compositions and method for treatment of chronic inflammatory diseases
- L11 ANSWER 2 OF 27 HCAPLUS COPYRIGHT 2009 ACS on STN
- TI Peptides and peptide mimetics to treat pathologies characterized by an inflammatory response
- L11 ANSWER 3 OF 27 HCAPLUS COPYRIGHT 2009 ACS on STN
- TI Use of lipid conjugates in the treatment of infection
- L11 ANSWER 4 OF 27 HCAPLUS COPYRIGHT 2009 ACS on STN
- TI Design of modified glycosaminoglycan-binding chemokines and other proteins for the treatment of inflammatory diseases
- L11 ANSWER 5 OF 27 HCAPLUS COPYRIGHT 2009 ACS on STN
- TI Methods for differentiating stem cells using a self-replicating neocentromeric artificial chromosome with chromatin domains expressing

transgenes for gene therapy

- L11 ANSWER 6 OF 27 HCAPLUS COPYRIGHT 2009 ACS on STN
- ${\tt TI}$ Polymer-modified bioactive synthetic chemokines, and methods for their manufacture and therapeutic use
- L11 ANSWER 7 OF 27 HCAPLUS COPYRIGHT 2009 ACS on STN
- TI Compositions treatment of chronic inflammatory diseases
- L11 ANSWER 8 OF 27 HCAPLUS COPYRIGHT 2009 ACS on STN
- TI Pharmaceutical compositions
- L11 ANSWER 9 OF 27 HCAPLUS COPYRIGHT 2009 ACS on STN
- TI Gene expression profiles and biomarkers for the detection of lung disease-related and other disease-related gene transcripts in blood
- L11 ANSWER 10 OF 27 HCAPLUS COPYRIGHT 2009 ACS on STN
- TI Gene expression profiles and biomarkers for the detection of depression-related and other disease-related gene transcripts in blood
- L11 ANSWER 11 OF 27 HCAPLUS COPYRIGHT 2009 ACS on STN
- TI Genes showing altered expression in lung cancer and their products and their use in diagnosis and treatment
- L11 ANSWER 12 OF 27 HCAPLUS COPYRIGHT 2009 ACS on STN
- TI Human tissue-specific housekeeping genes identified by expression profiling
- L11 ANSWER 13 OF 27 HCAPLUS COPYRIGHT 2009 ACS on STN
- TI Preparation of diaminothiadiazole dioxides and monoxides as CXC- and CC-chemokine receptor ligands
- L11 ANSWER 14 OF 27 HCAPLUS COPYRIGHT 2009 ACS on STN
- TI Preparation of amino acid derivatives in methods for the treatment of respiratory diseases and conditions with a selective iNOS inhibitor and a PDE inhibitor
- L11 ANSWER 15 OF 27 HCAPLUS COPYRIGHT 2009 ACS on STN
- TI Novel antagonists of MCP proteins for treating inflammatory, autoimmune and vascular diseases
- L11 ANSWER 16 OF 27 HCAPLUS COPYRIGHT 2009 ACS on STN
- TI Preparation of diaminocyclobutene-1,2-diones for combination treatments for chemokine-mediated diseases
- L11 ANSWER 17 OF 27 HCAPLUS COPYRIGHT 2009 ACS on STN
- TI Modular biochip arrays and their diagnostic or analytical uses and their preparation and uses
- L11 ANSWER 18 OF 27 HCAPLUS COPYRIGHT 2009 ACS on STN
- TI Genes that are differentially expressed during erythropoiesis and their diagnostic and therapeutic uses
- L11 ANSWER 19 OF 27 HCAPLUS COPYRIGHT 2009 ACS on STN
- TI Gene markers useful for detecting skin damage in response to ultraviolet radiation
- L11 ANSWER 20 OF 27 HCAPLUS COPYRIGHT 2009 ACS on STN
- TI Screening methods to identify compounds that modulate a gene expression response of a cell to ultraviolet radiation exposure

- L11 ANSWER 21 OF 27 HCAPLUS COPYRIGHT 2009 ACS on STN
- TI Modulation of airway responsiveness by anionic and cationic polyelectrolyte substances
- L11 ANSWER 22 OF 27 HCAPLUS COPYRIGHT 2009 ACS on STN
- TI Gene probes used for genetic profiling in healthcare screening and planning
- L11 ANSWER 23 OF 27 HCAPLUS COPYRIGHT 2009 ACS on STN
- TI Gene probes used for genetic profiling in healthcare screening and planning
- L11 ANSWER 24 OF 27 HCAPLUS COPYRIGHT 2009 ACS on STN
- TI Enhanced channelling of sulphate through a rapidly exchangeable sulphate pool in response to stimulated glycosaminoglycan synthesis in pancreatic epithelial cells
- L11 ANSWER 25 OF 27 HCAPLUS COPYRIGHT 2009 ACS on STN
- TI Polycations increase the efficiency of adenovirus-mediated gene transfer to epithelial and endothelial cells in vitro
- L11 ANSWER 26 OF 27 HCAPLUS COPYRIGHT 2009 ACS on STN
- TI Cationic proteins increase the permeability of cultured rabbit tracheal epithelial cells: Modification by heparin and extracellular calcium
- L11 ANSWER 27 OF 27 HCAPLUS COPYRIGHT 2009 ACS on STN
- TI Inhibition by salbutamol of the proliferation of human airway smooth muscle cells grown in culture
- => d 111 1 6 7 8 14 15 21 25 26 ti abs bib
 YOU HAVE REQUESTED DATA FROM FILE 'HCAPLUS' CONTINUE? (Y)/N:y
- L11 ANSWER 1 OF 27 HCAPLUS COPYRIGHT 2009 ACS on STN
- TI Pharmaceutical compositions and method for treatment of chronic inflammatory diseases
- AB This invention defines novel compns. that can be used for clin. treatment of a class of chronic inflammatory diseases. Increased generation of carbonyl substances, namely aldehydes and ketones, occurs at sites of chronic inflammation and is common to the etiologies of all of the clin. disorders addressed herein. Such carbonyl substances are cytotoxic and addnl. serve to perpetuate and disseminate the inflammatory process. This invention defines use of compns., the orally administered required primary agents of which are primary amine derivs. of benzoic acid capable of covalently reacting with the carbonyl substances. P-Aminobenzoic acid is an example of the required primary agent of the present invention. PABA has a small mol. weight, is water-soluble, has a primary amine group which reacts with carbonyl-containing substances and is tolerated by the body in relatively high dosages for extended periods. The method includes administration of a composition comprising: (1) an orally consumed therapeutically effective amount of at least one required primary agent; (2) at least one required previously known medicament co-agent recognized as effective to treat a chronic inflammatory disease addressed herein administered to the mammalian subject via the oral route; and (3) one or more addnl. orally consumed required co-agent selected from the group consisting of antioxidants, vitamins, metabolites at risk of depletion,

sulfhydryl co-agents, co-agents which may facilitate glutathione activity and nonabsorbable primary amine polymeric co-agents; so as to-produce an additive or synergistic physiol. effect of an anti-inflammatory nature.

AN 2008:1156137 HCAPLUS <<LOGINID::20090514>>

DN 149:409732

TI Pharmaceutical compositions and method for treatment of chronic inflammatory diseases

IN Shapiro, Howard K.

PA USA

SO U.S. Pat. Appl. Publ., 35pp., Cont.-in-part of U.S. Ser. No. 924,945.

DT Patent

LA English

FAN.CNT 5

T T 71.4 *	0111				
	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
ΡI	US 20080234380	A1	20080925	US 2008-70518	20080220 <
	US 20050090553	A1	20050428	US 2004-924945	20040824 <
PRAI	US 1992-906909	B2	19920630	<	
	US 1994-241603	В2	19940511	<	
	US 1997-814291	B2	19970310	<	
	US 2000-610073	B2	20000705	<	
	US 2004-924945	A2	20040824		

- L11 ANSWER 6 OF 27 HCAPLUS COPYRIGHT 2009 ACS on STN
- TI Polymer-modified bioactive synthetic chemokines, and methods for their manufacture and therapeutic use
- AΒ The invention relates to polymer-modified bioactive synthetic chemokines and to methods for their production and use. The bioactive synthetic chemokines of the invention comprises a polymer modified polypeptide chemokine backbone. The compds. and methods or the invention are useful for the treatment of disorders involving naturally occurring chemokines, such as for the treatment of HIV and AIDS related disorders and for the treatment of asthma, allergic rhinitis, atopic dermatitis, atheroma/atherosclerosis, organ transplant rejection, and rheumatoid arthritis (no data). Thus, solid-phase peptide synthesis was used to prepare an N- and a C-terminal fragment of Rantes. A thioester-generating resin was used for the N-terminal peptide and a standard phenylacetamidomethyl resin for the C-terminal peptide. Full-length (modified) Rantes peptides were produced by natural chemical ligation of the two fragments. Rantes derivs. with a fatty acyl group attached to the N-terminus or to a lysine side chain, as well as such derivs. containing nonnatural amino acids at various positions in the peptide chain, were prepared and tested for their ability to inhibit HIV envelope-mediated cell fusion and viral infection of a cell line.
- AN 2005:370952 HCAPLUS <<LOGINID::20090514>>

DN 142:435737

- TI Polymer-modified bioactive synthetic chemokines, and methods for their manufacture and therapeutic use
- IN Bradburne, James A.; Kochendoerfer, Gerd G.; Wilken, Jill G.

PA USA

SO U.S. Pat. Appl. Publ., 83 pp., Cont.-in-part of U.S. Provisional Ser. No. 217,683.

CODEN: USXXCO

DT Patent

LA English

FAN.CNT 3

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE				
ΡI	US 20050089970	A1	20050428	US 2003-332039	20030106 <				
	WO 2002004015	A1	20020117	WO 2001-US21933	20010712 <				

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WO 2002004015
                                20030807
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             LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL,
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             UZ, VN, YU, ZW
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     JP 2007302667
                         Α
                               20071122
                                           JP 2007-125054
                                                                  20070509 <--
PRAI US 2000-217683P
                         Ρ
                                20000712 <--
     WO 2001-US21933
                         W
                                20010712 <--
                         A3
     JP 2002-508469
                                20010712 <--
    ANSWER 7 OF 27 HCAPLUS COPYRIGHT 2009 ACS on STN
L11
    Compositions treatment of chronic inflammatory diseases
ΤI
     This invention defines novel compns. that can be used for clin. treatment
AΒ
     of a class of chronic inflammatory diseases. Increased generation of
     carbonyl substances, aldehydes and ketones, occurs at sites of chronic
     inflammation and is common to the etiologies of all of the clin. disorders
     addressed herein. Such carbonyl substances are cytotoxic and addnl. serve
     to perpetuate and disseminate the inflammatory process. This invention
     defines use of compns., the orally administered required primary agents of
     which are primary amine derivs. of benzoic acid capable of reacting with
     the carbonyl substances. P-Aminobenzoic acid (or PABA) is an example of
     the required primary agent of the present invention. PABA has a small
     mol. weight, is water soluble, has a primary amine group which reacts with
     carbonyl-containing substances and is tolerated by the body in relatively high
     dosages for extended periods. The method of the present invention
     includes administration of a composition comprising: (1) an orally consumed
     primary agent; (2) a previously known medicament co-agent recognized as
     effective to treat a chronic inflammatory disease addressed herein
     administered to the mammalian subject via the oral route, other systemic
     routes of administration or via the topical route; and (3) optionally 1 or
     more addnl. orally consumed co-agent selected from the group consisting of
     antioxidants, vitamins, metabolites at risk of depletion, sulfhydryl
     co-agents, co-agents which may facilitate glutathione activity and
     nonabsorbable primary amine polymeric co-agents, so as to produce an
     additive or synergistic physiol. effect of an anti-inflammatory nature.
ΑN
     2005:369133 HCAPLUS <<LOGINID::20090514>>
DN
     142:435774
ΤI
     Compositions treatment of chronic inflammatory diseases
IM
     Shapiro, Howard K.
PA
SO
     U.S. Pat. Appl. Publ., 44 pp., Cont.-in-part of U.S. Ser. No. 610,073,
     abandoned.
     CODEN: USXXCO
DT
     Patent
LA
     English
FAN.CNT 5
     PATENT NO.
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                                DATE
                                           APPLICATION NO.
                                                                   DATE
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     US 20050090553
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PΙ
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                        В2
     US 1997-814291
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                        В2
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                        A2
                                20040824
     US 2004-924945
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MARPAT 142:435774

OS

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L11 ANSWER 8 OF 27 HCAPLUS COPYRIGHT 2009 ACS on STN
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TI Pharmaceutical compositions

AB The present invention relates to pharmaceutical compns. which are useful in the treatment of diseases where excess mucus is present in the respiratory tract, such as cystic fibrosis and chronic obstructive pulmonary disease

In particular, the invention relates to pharmaceutical compns. for administration by pulmonary inhalation. Thus, in a first aspect of the present invention, a composition for assisting mucus clearance is provided, the composition comprising one or more mucoactive agents for reducing crosslinking within the mucus; for diluting the mucus; and/or for digesting naked DNA and cell debris within the mucus. Preferably, the composition according to the invention further has the effect of reducing inflammation. In one embodiment of the present invention, the composition comprises one or more mucoactive agents together with an addnl. active agent such as an anti-inflammatory agent. In a particularly preferred embodiment of the present invention, the mucoactive agent for reducing crosslinking is a glycosaminoglycan such as heparin. A further group of mucoactive agents capable of assisting mucus clearance are amino acids. Acetylcysteine (NAC) and the acetylcysteine salt derivative Nacystelyn (or NAL) are also effective mucoactive agents which are suitable for inclusion in the compns. of the present invention.

AN 2005:259852 HCAPLUS <<LOGINID::20090514>>

DN 142:329858

TI Pharmaceutical compositions

IN Morton, David; Ganderton, David; Staniforth, John; Kamlag, Yorick

PA Vectura Limited, UK

SO PCT Int. Appl., 60 pp. CODEN: PIXXD2

DT Patent

LA English

FAN.CNT 1

PΙ

PA:	TENT :	NO.			KIND DATE						ICAT							
					A2 20050324 A3 20050616				,									
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CA	SN, TD, TG AU 2004271778 CA 2538399 EP 1663151				A1	20050324			1	AU 2004-271778 CA 2004-2538399 EP 2004-768478					20040915 <			<
BR CN JP SG KR MX ZA	R: AT, BE, CH, IE, SI, LT,					DK, FI,	ES, RO, 2006 2006 2007 2008 2006 2006 2007	FR, MK, 1114	EP 2004-768478 GB, GR, IT, LI, LU, NL, CY, AL, TR, BG, CZ, EE, BR 2004-14425 CN 2004-80032679 JP 2006-525902 SG 2008-6902 KR 2006-705166 MX 2006-2952 ZA 2006-2748						SE, HU, 20 20 20 20 20 20	MC, PL, 0040 0040 0040 0040 0060 0060	PT, SK, 915 < 915 < 915 <	HR < < < < <

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US 20070065373 A1 20070322 US 2006-571184 20060717 <--
PRAI GB 2003-21611
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    GB 2003-27723
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    WO 2004-GB3932
                               20040915
             THERE ARE 7 CITED REFERENCES AVAILABLE FOR THIS RECORD
RE.CNT 7
             ALL CITATIONS AVAILABLE IN THE RE FORMAT
L11 ANSWER 14 OF 27 HCAPLUS COPYRIGHT 2009 ACS on STN
    Preparation of amino acid derivatives in methods for
    the treatment of respiratory diseases and conditions with a selective iNOS
    inhibitor and a PDE inhibitor
    The invention claims a combination of an iNOS blocker and a
AΒ
    phosphodiesterase (PDE) inhibitor or their pharmaceutically-acceptable
    salts or prodrugs for the prevention and treatment of respiratory diseases
    or conditions. The iNOS inhibitors include amino acids
    HN: CMeNHCH2CHRSCH2CH(NH2)CO2H (R = alkyl, cycloalkyl, hydroxyalkyl, or
    haloalkyl). Thus, 2S-amino-6-[(1-iminoethyl)amino]-N-(1H-tetrazol-5-
    yl)hexanamide dihydrochloride (NN) was prepared and shown to be a more
    potent i-NOS inhibitor (IC50 = 21.4 \muM) than
    2S-amino-6-[(1-iminoethyl)amino]hexanamide (NIL amide) or NIL
    dimethylamide. NN is a nicely crystalline product, in contrast to NIL which is
    a glass and thus difficult to handle.
    2003:931174 HCAPLUS <<LOGINID::20090514>>
ΑN
DN
    140:16957
ΤI
    Preparation of amino acid derivatives in methods for
    the treatment of respiratory diseases and conditions with a selective iNOS
    inhibitor and a PDE inhibitor
ΙN
    Manning, Pamela T.
PA
    Pharmacia Corporation, USA
SO
    PCT Int. Appl., 245 pp.
    CODEN: PIXXD2
DT
    Patent
    English
LA
FAN.CNT 1
    PATENT NO.
                     KIND DATE
                                          APPLICATION NO.
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    WO 2003097050 A2 20031127
WO 2003097050 A3 20040617
                                          WO 2003-US15464
PΙ
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    CA 2484654
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    US 20040087653
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                                          EP 2003-753056
    EP 1505972
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                                           BR 2003-10061
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    MX 2004011335
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                                          MX 2004-11335
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                        Ρ
PRAI US 2002-381056P
                               20020516 <--
    WO 2003-US15464
                         W
                               20030516 <--
OS
    MARPAT 140:16957
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RE.CNT 17 THERE ARE 17 CITED REFERENCES AVAILABLE FOR THIS RECORD

ALL CITATIONS AVAILABLE IN THE RE FORMAT

- L11 ANSWER 15 OF 27 HCAPLUS COPYRIGHT 2009 ACS on STN
- TI Novel antagonists of MCP proteins for treating inflammatory, autoimmune and vascular diseases
- AB Novel antagonists of MCP proteins, in particular of MCP-1 protein, can be obtained by generating MCP mutants whose GAG binding site, located at the N-terminal of MCP proteins, is eliminated following non-conservative substitutions. Compds. prepared in accordance with the present invention can be used in the treatment or prevention of diseases related to an undesirable activity of MCP proteins such, such as inflammatory disease, autoimmune diseases, vascular diseases, and cancer. MCP-1WT*2A, human mature MCP-1 with isoleucine substituted for the methionine at position 64 and two alanines replacing the arginine and lysine at positions 18 and 19, was prepared in Escherichia coli. The mutant protein showed protective activity in animal models of delayed contact hypersensitivity, lung fibrosis, lung inflammation, and asthma.
- AN 2003:818453 HCAPLUS <<LOGINID::20090514>>
- DN 139:302042
- TI Novel antagonists of MCP proteins for treating inflammatory, autoimmune and vascular diseases
- IN Proudfoot, Amanda; Kosco-Vilbois, Marie; Handel, Tracy
- PA Applied Research Systems Ars Holding N.V., Neth. Antilles
- SO PCT Int. Appl., 71 pp. CODEN: PIXXD2
- DT Patent
- LA English
- FAN.CNT 1

FAN.		rent :	NO.			KIND DATE					APPL	ICAT	ION :	DATE					
ΡI	WO	2003084993			A1 20031016			WO 2003-EP50097						20030409 <					
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	AU	2003240765			A1 20031020				AU 2003-240765						20030409 <				
	EΡ	1495	050			A1 20050			0112	EP 2003-730178						20030409 <			
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		1665						2005	0907				8130						
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	ZA	2004	0090	62		A		2005	1109		ZA 2	004-	9062			2	0041	109	<
	US	2007	0004	906		A1		2007	0104		US 2	005-	5106	58		2	0050	518	<
	US	7425	324			В2		2008	0916										
PRAI	US	2002	-371	442P		P		2002	0410	0410 <									
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- RE.CNT 6 THERE ARE 6 CITED REFERENCES AVAILABLE FOR THIS RECORD ALL CITATIONS AVAILABLE IN THE RE FORMAT
- L11 ANSWER 21 OF 27 HCAPLUS COPYRIGHT 2009 ACS on STN
- TI Modulation of airway responsiveness by anionic and cationic polyelectrolyte substances

- To elucidate the effects of anionic and cationic polyelectrolyte substance AR on bronchoconstriction, we examined the serial changes in respiratory resistance (Rrs) in ovalbumin-sensitized guinea pigs after antigen exposure with or without preinhalation of low-mol.-weight heparin, poly-1-glutamic acid, poly-1-lysine and dextran with or without oral intake of dalteparin. Both immediate and late responses after antigen exposure were significantly decreased after pretreatment with inhaled low-mol.-weight heparin and poly-l-glutamic acid compared with saline alone. The late response was significantly decreased after pretreatment with oral dalteparin. Both low-mol.-weight heparin and poly-1-glutamic acid significantly decreased the airway response to methacholine in sensitized guinea pigs. In sensitized guinea pigs, the airway response to methacholine was significantly increased after pretreatment with inhaled poly-l-lysine. Pretreatment with inhaled low-mol.-weight heparin before poly-l-lysine exposure significantly suppressed the airway hyperresponsiveness after inhaled poly-1-lysine. These findings indicated that the "cationic-anionic interaction" plays an important role in airway responsiveness.
- AN 2002:2418 HCAPLUS <<LOGINID::20090514>>
- DN 136:338615
- TI Modulation of airway responsiveness by anionic and cationic polyelectrolyte substances
- AU Yahata, Tomoyuki; Nishimura, Yoshihiro; Maeda, Hitoshi; Yokoyama, Mitsuhiro
- CS Department of Internal Medicine, Division of Cardiovascular and Respiratory Medicine, Kobe University Graduate School of Medicine, Kobe, Chuo-ku, 650-0017, Japan
- SO European Journal of Pharmacology (2002), 434(1-2), 71-79 CODEN: EJPHAZ; ISSN: 0014-2999
- PB Elsevier Science B.V.
- DT Journal
- LA English
- RE.CNT 39 THERE ARE 39 CITED REFERENCES AVAILABLE FOR THIS RECORD ALL CITATIONS AVAILABLE IN THE RE FORMAT
- L11 ANSWER 25 OF 27 HCAPLUS COPYRIGHT 2009 ACS on STN
- TI Polycations increase the efficiency of adenovirus-mediated gene transfer to epithelial and endothelial cells in vitro
- AΒ Recombinant adenoviruses are being developed for gene therapy for cystic fibrosis and other lung diseases, and for prevention and treatment of vascular thrombosis. A major limitation to the clin. utility of adenoviruses is the low efficiency of gene transfer achieved in vivo. In addition, little is known about the initial interactions between adenoviruses and the target cell. To address the hypothesis that the neg. charge presented by membrane glycoproteins reduces the efficiency of adenovirus-mediated gene transfer, primary cultures of human airway, Madin-Darby canine kidney cells, an immortalized cystic fibrosis airway epithelial cell line, and primary cultures of sheep pulmonary artery endothelium were infected with recombinant adenovirus containing the E. coli lacZ reporter gene (Ad2 β gal2) in the presence of various polyions. For each cell type, adsorption of $Ad2\beta gal2$ in the presence of the polycations polybrene, protamine, DEAE-dextran, and poly-L-lysine significantly increased the percentage of cells that express lacZ. The polyanion heparin did not significantly alter gene transfer efficiency, but completely abrogated the effects of polycations. These data provide evidence that neg. charged moieties on the cell surface reduce the efficiency of adenovirus-mediated gene transfer, and that alteration of the charge interaction between adenoviruses and the cell surface may improve the potential clin. application of these vectors.

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AN 1997:82048 HCAPLUS <<LOGINID::20090514>>
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- DN 126:195011
- OREF 126:37511a,37514a
- TI Polycations increase the efficiency of adenovirus-mediated gene transfer to epithelial and endothelial cells in vitro
- AU Arcasoy, S. M.; Latoche, J. D.; Gondor, M.; Pitt, B. R.; Pilewski, J. M.
- CS Dep. Med., Univ. Pittsburgh Sch. Med., Pittsburgh, PA, USA
- SO Gene Therapy (1997), 4(1), 32-38 CODEN: GETHEC; ISSN: 0969-7128
- PB Stockton
- DT Journal
- LA English
- L11 ANSWER 26 OF 27 HCAPLUS COPYRIGHT 2009 ACS on STN
- TI Cationic proteins increase the permeability of cultured rabbit tracheal epithelial cells: Modification by heparin and extracellular calcium
- Airway inflammation is a consistent finding in asthma, and AΒ increased amts. of eosinophil-derived cationic proteins are present in bronchoalveolar lavage fluid from asthmatic subjects. Tracheal instillation of a variety of naturally occurring and synthetic cationic proteins has been shown to induce airway hyperresponsiveness in animal models. Cationic proteins may alter the barrier function of airway epithelium, allowing increased access of agonists to underlying nerves and airway smooth muscle. To examine the effect of cationic proteins on airway epithelial cell function, rabbit tracheal epithelial cells were isolated and cultured on collagen-coated filter membranes. Both apical and basolateral exposure of the cell cultures to poly-L-lysine and poly-L-arginine decreased transepithelial elec. resistance (Rt) over 60 min. There were no discernable light microscopic changes in the morphol. of the cultures at 60 min after poly-L-lysine exposure, but permeability to mannitol was increased compared to controls. for the critical role of cationic charge included the following observations:. (1) Poly-L-aspartate, an anionic polyamino acid, had no significant effect on Rt, and (2) the addition of heparin prior to the addition of poly-L-lysine blocked the reduction in Rt. Furthermore, when applied after poly-L-lysine addition, heparin reversed the decrease in Rt in a time-dependent fashion. Increasing the [Ca2+] in the medium from 1 to 10 mM resulted in attenuation of the response to polycation addition Cationic proteins may alter the barrier properties of airway epithelium and the cationic charge may be a crucial factor. This alteration is not an 'all-or-none' phenomenon, since subsequent addition of heparin resulted in a reversal of the effect. While the precise mechanisms responsible for these observations remain to be elucidated, cationic proteins may be modifying the interaction of extracellular calcium with tight junctions thereby resulting in increased permeability. The barrier function of the epithelium may be perturbed in asthma and a variety of other airway diseases through the presence of cationic proteins derived from inflammatory cells within the airway lumen and/or the subepithelium.
- AN 1996:277186 HCAPLUS <<LOGINID::20090514>>
- DN 124:332524
- OREF 124:61409a,61412a
- TI Cationic proteins increase the permeability of cultured rabbit tracheal epithelial cells: Modification by heparin and extracellular calcium
- AU Uchida, Derek A.; Irvin, Charles G.; Ballowe, Clark; Larsen, Gary; Cott, Gary R.
- CS School Medicine, University Colorado, Denver, CO, USA
- SO Experimental Lung Research (1996), 22(1), 85-99 CODEN: EXLRDA; ISSN: 0190-2148

PB Taylor & Francis DT Journal LA English